**Case Report** 

# **Robotic Strong's Procedure for Superior Mesenteric**

**Artery Syndrome** 

Carolyn Judge\*, Marian-Joy Baluyot, Cyril Harfouche, Derek Benham, Kyle Gadbois and Gordon Wisbach

Department of General Surgery, Navy Medicine Readiness and Training Command, USA

### Abstract

Superior Mesenteric Artery Syndrome (SMAS) is a rare condition caused by vascular compression of the duodenum by the superior mesenteric artery. We present the case of a 24-year-old female with a BMI of 18.96 who presented with post-prandial abdominal pain, emesis, and failure to thrive. Upper endoscopy demonstrated partial obstruction of the duodenum at the level of the third portion by a pulsatile mass. Subsequent UGI and CT redemonstrated this finding and revealed an aortomesenteric angle of 18, both consistent with a diagnosis of SMAS. After failing conservative efforts to gain weight over a 6-month period, the decision was made to proceed with surgery. A Strong's procedure was chosen due to its minimally invasive nature. It is defined by division of the suspensory ligament of the duodenum to reposition the bowel to the right of the aorta such that it no longer lies within the aortomesenteric angle. It does not violate the integrity of the gastrointestinal tract thereby avoiding the complications of anastomosis. Robotic Strong's procedures are safe and effective for the treatment of SMAS in part because of the precision and accuracy of the division of the suspensory ligament of the duodenum as well as circumferential dissection of the superior mesenteric axis and aortomesenteric space, far more challenging with laparoscopic instruments.

Keywords: Strong's Procedure; Robotic strong's procedure; Superior mesenteric artery syndrome; Duodenal obstruction; Wilkie's syndrome

## Introduction

SMAS is defined by mechanical obstruction of the 3<sup>rd</sup> portion of the duodenum by the superior mesenteric artery [1]. It is thought to be caused by rapid weight loss resulting in loss of the intraabdominal fat pad between the SMA and the aorta thereby acutely decreasing the Aortomesenteric Angle (AMA) and compressing the bowel as it traverses the space [1]. The syndrome was first described by Dr. Rokitansky in 1842 [2]. In a case series of over 75 patients, Dr. Wilkie further described the anatomy and physiology of this disease for which it commonly bears his namesake [3]. It has also been referred to as chronic duodenal ileus and cast syndrome due to the association with prolonged immobility in patients with body casts [4].

Conservative therapy should be attempted prior to surgical intervention, but has historically had poor success in adult cohorts [5]. The duodenojejunostomy, first described by Dr. Stavely in 1908, is the most commonly employed procedure [6]. An alternative is Strong's procedure wherein the duodenum is mobilized by division of the ligament of Treitz allowing it to fall inferiorly and relieving the obstruction without violating gastrointestinal continuity [7]. Laparoscopic duodenojejunostomy and Strong's procedures have been demonstrated to be safe and effective [8,9]. Robotic duodenojejunostomy and Strong's procedures have also been successful in the treatment of SMAS [10-12]. The robotic platform combines the

Citation: Judge C, Baluyot M-J, Harfouche C, Benham D, Gadbois K, Wisbach G. Robotic Strong's Procedure for Superior Mesenteric Artery Syndrome. Am J Surg Case Rep. 2022;3(5):1049.

Copyright: © 2022 Carolyn Judge

Publisher Name: Medtext Publications LLC

Manuscript compiled: Dec 21st, 2022

\*Corresponding author: Carolyn G Judge, Department of General Surgery, Navy Medicine Readiness and Training Command, San Diego, 291 Awakea Rd, Kailua, HI, 96734, USA, Tel: +1-203-247-7736; E-mail: Carolyn.g.judge.mil@mail.mil

benefits of minimally invasive technique and unparalleled dissection, which may improve the safety and efficacy of Strong's procedure. We present the case of successful surgical management of SMAS by robotic-assisted laparoscopic Strong's Procedure.

#### **Case Presentation**

Pre-operative EGD demonstrated evidence of duodenal compression by what was observed to be a pulsatile mass. Subsequent UGI captured the contrast transit to the third portion of the duodenum, but with the majority remaining in the stomach. CT images showed an AMA of 18 degrees as well as vascular compression of the third portion of the duodenum, gastric dilation, and a focal narrowing of the renal vein as it passed between the aorta and the superior mesenteric artery (Figure 1-3).

Given history and clinical findings concerning for superior mesenteric artery syndrome in the setting of failed conservative efforts to regain weight over a 6-month period, and with consideration for the patient's strong aversion to TPN and enteral feeding, the decision was made to pursue surgical management. A robotic Strong's



Figure 1: UGI demonstrating a small amount of contrast in the third portion of the duodenum with the majority remaining in the stomach.



**Figure 2 and 3**: CT images demonstrating an aortomesenteric angle of 18 degrees as well as vascular compression of the third portion of the duodenum, gastric dilation, and a focal narrowing of the renal vein within the aortomesenteric angle.

procedure was chosen due to the proven success in the treatment of this syndrome as well as desire to avoid violation of the continuity of the GI tract, immediate anastomotic risks, and long-term physiologic risks of a duodenojejunostomy given her age.

Exposure of the ligament of Treitz, which was noted to be short, was accomplished using a hammock technique. This involved passage of <sup>1</sup>/<sub>2</sub> inch penrose drains, which were then secured by endoloops and fixed to the abdominal wall *via* right and left upper quadrant stab incisions using a trans-fascial suture passer. Once satisfactory exposure achieved, upper endoscopy was performed redemonstrating the known partial obstruction at the level of the third portion of the duodenum. Of note, there was no evidence of duodenal malrotation.

Dissection proceeded in a clockwise fashion, using cautery too circumferentially to dissect the superior mesenteric axis, the aortomesenteric angle and to divide the ligament of Treitz as well as to mobilize the third and fourth portions of the duodenum (Figure 4). Firefly technology using indocyanine green was used to confirm vascular anatomy including the locations the superior mesenteric artery and vein. With the ligament of Treitz divided and the duodenum mobilized, the jejunum was further mobilized, and tension-free loops were passed behind the SMA allowing the duodenum to fall inferiorly such that it was no longer positioned in the aortomesenteric angle.

Satisfied with the complete release of the suspensory ligament and all potential pathologic attachments, the procedure was concluded. There were no complications. Post-operative UGI demonstrated passage of contrast into the third portion of the duodenum consistent with resolution of obstruction (Figure 5).

## **Discussion**

SMAS is caused by vascular compression of the third portion of the duodenum by the SMA due to loss of the inferior mesenteric fat pad in the setting of rapid weight loss [1]. The fat pad sits beneath the SMA takeoff from the aorta and typically maintains the AMA between 38 and 56 degrees [1,13]. When less than 25 degrees and associated with a decrease in the Aortomesenteric Distance (AMD) from 10 mm-20 mm to 2 mm-8 mm, it is diagnostic of SMAS [1].

While there are many conditions which might precipitate this condition, they usually have in common nausea, anorexia, and pain which perpetuate a cycle of weight loss and worsening pathology [1].

There may be anatomic variations which predispose to the condition such as high insertion of the LOT, low insertion of the SMA, adhesions causing duodenal compressions, intestinal malrotation, or local pathology (neoplastic growth in the mesenteric root, dissecting aortic aneurysm [14,15]. Unlike familial megaduodenum, included in the



Figure 4: Hammock technique used to expose the ligament of Treitz.





differential, SMAS is not caused by hypertrophy, but rather presents as a true mechanical obstruction of an otherwise normal area of bowel [1]. There has been discussion of a functional obstruction due to a segmental motility disorder, but this is not strongly supported in surgical literature [16].

SMAS is rare with an estimated incidence of 0.3% [17]. This disease most commonly occurs in females and young adults [3] and is strongly associated with psychosocial conditions including eating disorders [1]. Patients may present acutely following trauma or burns, or with more chronic symptoms due to cancer, chronic inflammatory states such as AIDS, or anorexia [1]. Patients commonly note abdominal pain, which is relieved in a prone or left lateral recumbent position both of which would release tension on the small bowel mesentery, widening the AMA and temporarily alleviating the obstruction [1]. Conversely, the pain may be noted to worsen while supine due to closure of the angle [13]. Significant obstruction may cause bilious emesis, weight loss of 33%-55% of body weight, and electrolyte abnormalities [1,18].

Barium or CT studies confirm diagnosis by demonstrating abrupt cutoff of contrast passage at the level of the  $3^{rd}$  portion of the duodenum [13]. These studies may further demonstrate delayed

gastric emptying, antiperistaltic flow, and/or a vertical band-like defect across the compressed area [13]. Ultrasound has also been shown to be highly successful identifying the AMA and AMD in the diagnosis of SMAS [19]. Upper gastrointestinal endoscopy should be considered to rule out other causes of obstruction [20].

Conservative therapy to address the weight loss, resuscitate the patients, and promote nutrition either distally to the obstruction or in a liquid medium able to transit a partial obstruction, should be exhausted before considering surgical options [1,14]. Success of 14%-83% has been reported but is less likely when the duration of symptoms is greater than a month or when there has been no significant clinical response with six weeks of conservative therapy [21]. Interestingly, there is no data on how much weight must be regained to reconstitute the fat pad and alleviate symptoms [13].

Surgical options include the duodenojejunostomy [6], gastrojejunostomy [22], and Strong's procedure [7]. Infrarenal transposition of the SMA has also been reported but has not gained favor likely due to incompatibility with minimally invasive techniques [23]. The duodenojejunostomy is generally regarded as the preferred operation, with success rates of 80%-90% reported in the classically described open approach [24]. Duodenojejunostomy does, however, carry the risk of blind loop syndrome should the fourth portion of the duodenum not be divided [1]. Gastrojejunostomy may alleviate symptoms due to decompression of the stomach, but fails to address the pathologic distal obstruction and may cause blind loop syndrome and significant bile reflux [12,25]. Strong's procedure mobilizes the duodenum away outside of the AMA by division of the LOT [7], preserving the integrity of the GI tract and avoiding potential complications of anastomosis or physiological derangement. A failure rate of up to 25% has been reported and is thought to be due to short branches of the inferior pancreaticoduodenal artery restricting duodenal movement [1].

The major advantage of robotic technology in this operation is the potential for enhanced execution of the critical steps: recognition of the SMA vessels, accurate mobilization of the duodenum, and circumferential dissection of the SMA and aortomesenteric space. There is not enough data for comparative study to laparoscopic approach; however, the existing literature suggests minimal blood loss, short operative time, short hospital say, and early recovery [10-12]. The robotic approach is safe and effective in the treatment of SMAS [10-12] and should be considered in management of these patients.

#### **References**

- Merrett ND, Wilson RB, Cosman P, Biankin AV. Superior mesenteric artery syndrome: diagnosis and treatment strategies. J Gastrointest Surg. 2009;13(2):287-92.
- Rokitansky V. Superior mesenteric artery syndrome. Lehrb Pathol Anat. 1861;(3<sup>rd</sup> ed.):87.
- 3. Wilkie BP. Chronic duodenal ileus. Am J Med Sci. 1927;173:643-650.
- Hughes JP, McEntire JE, Setze TK. Cast syndrome: duodenal dilation or obstruction in a patient in a body cast, with review of the literature. Arch Surg. 1974;108(2):230-2.

- Reckler JM, Bruck HM, Munster AM, Curreri PW, Pruitt Jr BA. Superior mesenteric artery syndrome as a consequence of burn injury. J Trauma. 1972;12(11):979-85.
- Stavely AL. Acute and chronic gastromesenteric ileus with cure in a chronic case by duodenojejunostomy. Bull Johns Hopkins Hosp. 1908;19:252.
- Strong EK. Mechanics of arteriomesentric duodenal obstruction and direct surgical attack upon etiology. Ann Surg. 1958;148(5):725-30.
- Massoud WZ. Laparoscopic management of superior mesenteric artery syndrome. Int Surg. 1995;80(4):322-7.
- Gersin KS, Heniford BT. Laparoscopic duodenojejunostomy for treatment of superior mesenteric artery syndrome. JSLS. 1998;2(3):281-4.
- Ayloo SM, Choudhury N. Robotic revisional bariatric surgery: single-surgeon case series. Int J Med Robot. 2015;11(3):284-9.
- Bütter A, Jayaraman S, Schlachta C. Robotic duodenojejunostomy for superior mesenteric artery syndrome in a teenager. J Robot Surg. 2010;4(4):265-9.
- 12. Konstantinidis H, Charisis C, Kottos P. Robotic Strong's procedure for the treatment of superior mesenteric artery syndrome. Description of surgical technique on occasion of the first reported case in the literature. Int J Med Robot. 2018;14(1):e1876.
- Warncke ES, Gursahaney DL, Mascolo M, Dee E. Superior mesenteric artery syndrome: a radiographic review. Abdom Radiol (NY). 2019;44(9):3188-94.
- Welsch T, Büchler MW, Kienle P. Recalling superior mesenteric artery syndrome. Dig Surg. 2007;24(3):149-56.
- Kyslan K, Barla J, Stanislayova M. [Superior mesenteric artery (SMAS/AMS) syndrome and its management]. Rozhl Chir. 2008;87(5):255-8.
- Martin R, Khor TS, Vermeulen T, Hall J. Wilkies Syndrome may be due to poor motility. ANZ J Surg. 2005;75(11):1027.
- Nugent FW, Braasch JW, Epstein H. Diagnosis and surgical treatment of arteriomesenteric obstruction of the duodenum. JAMA. 1966;196(12):1091-3.
- Cohen LB, Field SP, Sachar DB. The superior mesenteric artery syndrome. The disease that isn't, or is it? J Clin Gastroenterol. 1985;7(2):113-116.
- Neri S, Signorelli SS, Mondati E, Pulvirenti D, Campanile E, Di Pino L, et al. Ultrasound imaging in diagnosis of superior mesenteric artery syndrome. J Intern Med. 2005;257(4):346-51.
- Valiathan G, Wani M, Lanker J, Reddy PK. A Case Series on Superior Mesenteric Artery Syndrome Surgical Management, Single Institution Experience. J Clin Diagn Res. 2017;11(8):PR01-PR03.
- Shin MS, Kim JY. Optimal duration of medical treatment in superior mesenteric artery syndrome in children. J Korean Med Sci. 2013;28(8):1220-5.
- 22. Lee CS, Mangla JC. Superior mesenteric artery compression syndrome. Am J Gastroenterol Springer Nat. 1978;70(2).
- 23. Pourhassan S, Grotemeyer D, Fürst G, Rudolph J, Sandmann W. Infrarenal transposition of the superior mesenteric artery: a new approach in the surgical therapy for Wilkie syndrome. J Vasc Surg. 2008;47(1):201-4.
- Raissi B, Taylor BM, Taves DH. Recurrent superior mesenteric artery (Wilkie's) syndrome: a case report. Can J Surg. 1996;39(5):410-6.
- Pottorf BJ, Husain FA, Hollis HW, Lin E. Laparoscopic management of duodenal obstruction resulting from superior mesenteric artery syndrome. JAMA Surg. 2014;149(12):1319-22.